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=> s 11 SAMPLE SEARCH INITIATED 13:27:51 FILE 'REGISTRY' SAMPLE SCREEN SEARCH COMPLETED -1 TO ITERATE

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=> s 11 full

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FILE COVERS 1907 - 12 Jun 2009 VOL 150 ISS 25 FILE LAST UPDATED: 11 Jun 2009 (20090611/ED) REVISED CLASS FIELDS (/NCL) LAST RELOADED: Feb 2009 USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Feb 2009

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=> d 13 scan

10 ANSWERS REGISTRY COPYRIGHT 2009 ACS on STN Naphth [2,3-b] oxirene-2,7-dione, 1a,7a-dihydro-1a-methyl-7a-(3,7,11,15tetramethyl-2-hexadecenyl)-, $[1aS-[1a\alpha,7a\alpha(2E,7S*,11S*)]]$ -(9CI)

C31 H46 O3 MF

Absolute stereochemistry. Double bond geometry as shown.

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):10

- L3 10 ANSWERS REGISTRY COPYRIGHT 2009 ACS on STN
- IN Naphth[2,3-b]oxirene-2,7-dione-2-180, 1a,7a-dihydro-7a-methyl-1a-(3,7,11,15-tetramethyl-2-hexadecenyl)-, (E)-(9CI)
- MF C31 H46 O3

Double bond geometry as shown.

- L3 10 ANSWERS REGISTRY COPYRIGHT 2009 ACS on STN
- IN Naphth[2,3-b]oxirene-2,7-dione, 1a,7a-dihydro-1a-methyl-7a-(3,7,11-trimethyl-2-dodecen-1-yl)-
- MF C26 H36 O3

- **PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**
- L3 10 ANSWERS REGISTRY COPYRIGHT 2009 ACS on STN

IN Naphth[2,3-b]oxirene-2,7-dione, 1a,7a-dihydro-1a-methyl-7a-(3,7,11,15tetramethyl-2-hexadecenyl)-, $[1aR-[1a\alpha,7a\alpha(2E,7R^*,11R^*)]]$ -(9CI)

ME C31 H46 O3

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

L3 10 ANSWERS REGISTRY COPYRIGHT 2009 ACS on STN

IN Naphth [2,3-b]oxirene-2,7-dione-2-180, 1a, 7a-dihydro-1a-methyl-7a-(3, 7, 11, 15-tetramethyl-2-hexadecenyl)-, (E)-(9CI)

MF C31 H46 O3

Double bond geometry as shown.

- L3 10 ANSWERS REGISTRY COPYRIGHT 2009 ACS on STN
- IN Naphth[2,3-b]oxirene-2,7-dione-1-180,

1a, 7a-dihydro-la-methyl-7a-(3, 7, 11, 15-tetramethyl-2-hexadecenyl)-,

[7a(2E,7R,11R)]-[partial]- (9CI)

ME C31 H46 O3

Absolute stereochemistry. Double bond geometry as shown.

L3 10 ANSWERS REGISTRY COPYRIGHT 2009 ACS on STN

IN Naphth[2,3-b]oxirene-2,7-dione, 1a,7a-dihydro-la-methyl-7a-(3,7,11,15-tetramethyl-2-hexadecenyl-1,2-t2)- (9CI)

MF C31 H44 O3 T2

L3 10 ANSWERS REGISTRY COPYRIGHT 2009 ACS on STN

IN Naphth[2,3-b]oxirene-2,7-dione-1-180,

la,7a-dihydro-la-methyl-7a-(3,7,11,15-tetramethyl-2-hexadecenyl)- (9CI)

MF C31 H46 O3

L3 10 ANSWERS REGISTRY COPYRIGHT 2009 ACS on STN

IN Naphth[2,3-b]oxirene-2,7-dione-1,2-1802,

1a,7a-dihydro-la-methyl-7a-(3,7,11,15-tetramethyl-2-hexadecenyl)-,

[7a(2E,7R,11R)]-[partial]- (9CI)

MF C31 H46 O3

Absolute stereochemistry. Double bond geometry as shown.

IN Naphth[2,3-b]oxirene-2,7-dione, 1a,7a-dihydro-1a-methyl-7a-(3,7,11,15-tetramethyl-2-hexadecen-1-yl)-

MF C31 H46 O3

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

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2607136 TREATMENT

L7 9 L6 AND TREATMENT

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L7 ANSWER 1 OF 9 ZCAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2002:667430 ZCAPLUS

DOCUMENT NUMBER: 137:195570

TITLE: Methods of treating chronic inflammatory diseases using carbonyl trapping agents

INVENTOR(S): Shapiro, Howard K.

PATENT ASSIGNEE(S): USA

SOURCE: U.S., 23 pp., Cont.-in-part of U.S. Ser. No. 473,786,

abandoned. CODEN: USXXAM

DOCUMENT TYPE: Patent LANGUAGE: English

FAMILY ACC. NUM. COUNT: 5

PATENT INFORMATION: 5

PATENT NO. KIND DATE APPLICATION NO. DATE

US 6444221 B1 20020903 US 1999-416120 19991012
PRIORITY APPLN. INFO:: US 1992-06909 B2 19920600 US 1995-473786 B2 19950607

OTHER SOURCE(S): MARPAT 137:195570

AB These and other objects of this invention are achieved by providing a novel method and compns. for the clin. treatment of chronic inflammatory diseases. This invention involves use of systemically administered compns. Which include primary amine derivs. of benzoic acid as carbonyl trapping agents. These primary therapeutic agents act by chemical binding to and sequestering the aldehyde and/or ketone products of lipid peroxidn. Increased levels of lipid peroxidn. have been repeatedly demonstrated as a part of the non-enzymic "inflammatory cascade" process which underlies the secondary eticl. of chronic inflammatory diseases. P-Aminobenzoic acid (or PABA) is an example of the primary therapeutic agent of the present invention. PABA has a small mol. weight, is water

soluble,

has a primary amine group that reacts with carbonyl-containing metabolites under physiol. conditions and is tolerated by the body in relatively high dosages and for extended periods. The carbonyl sequestering agents are used in combination with at least one co-agent to produce an addnl. beneficial physiol. effect of an anti-inflammatory nature. Such compns. are administered systemically entirely via the oral route. Co-agents of the present invention include anti-oxidants and free radical trapping compds. (e.g., a-tocopherol), compds. having indirect anti-oxidant activity (e.g., selenium), vitamins (e.g., pyridoxine HCl), compds. which facilitate kidney drug elimination (e.g., glycine), metabolites at risk of depletion (e.g., pantothenic acid), sulfhydryl containing chems. (e.g., methionine), compds. which facilitate quitathione activity (e.g., ..., N-acetylcysteine), and non-absorbable polyamine co-agents (e.g., chitosan).

IT 25486-55-9, Vitamin K1 oxide

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(methods of treating chronic inflammatory diseases using primary amine derivs. of benzoic acid as carbonyl trapping agents and combination with other agents)

RN 25486-55-9 ZCAPLUS

CN Naphth[2,3-b]oxirene-2,7-dione, 1a,7a-dihydro-1a-methyl-7a-(3,7,11,15-tetramethyl-2-hexadecen-1-yl)- (CA INDEX NAME)

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 2 OF 9 ZCAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1990:511418 ZCAPLUS

DOCUMENT NUMBER: 113:111418
ORIGINAL REFERENCE NO.: 113:18777a,18780a

TITLE: Vitamin K1 2,3-epoxide and quinone reduction:

mechanism and inhibition

AUTHOR(S): Preusch, Peter C.; Smalley, David M.

CORPORATE SOURCE: Dep. Chem., Univ. Akron, Akron, OH, 44325, USA

SOURCE: Free Radical Research Communications (1990), 8(4-6), 401-15

CODEN: FRRCEX; ISSN: 8755-0199
DOCUMENT TYPE: Journal

LANGUAGE: English

The chemical and enzymic pathways of vitamin K1 epoxide and quinone reduction have been investigated. Na borohydride treatment resulted in carbonyl reduction generating relatively stable compds. that did not proceed to quinone in the presence of base. NAD(P)H:quinone oxidoreductase (DT-diaphorase) reduction of vitamin K to the hydroguinone was a significant process in intact microsomes, but 1/5th the rate of the dithiothreitol (DTT)-dependent reduction No evidence was found for DT-diaphorase catalyzed reduction of vitamin K1 epoxide, nor was it capable of mediating transfer of electrons from NADH to the microsomal epoxide reducing enzyme. Purified diaphorase reduced detergent-solubilized vitamin K1 10-5 as rapidly as it reduced dichlorophenylindophenol (DCPIP). Reduction of 10 μM vitamin K1 by 200 µM NADH was not inhibited by 10 µM dicoumarol, whereas DCPIP reduction was fully inhibited. In contrast to vitamin K3 (menadione), vitamin K1 (phylloquinone) did not stimulate microsomal NADPH consumption in the presence or absence of dicoumarol. DTT-dependent vitamin K epoxide reduction and vitamin K reduction were shown to be mutually inhibitory reactions, suggesting that both occur at the same enzymic site. On this basis, a mechanism for reduction of the quinone by thiols is proposed. Both the DTT-dependent reduction of vitamin K1 epoxide and quinone, and the reduction of DCPIP by purified DT-diaphorase were inhibited by dicoumarol, warfarin, lapachol, and sulfaquinoxaline.

T 25486-55-9, Vitamin K1 2,3-epoxide RL: RCT (Reactant); RRCT (Reactant or reagent) (reduction of, by sodium borohydride and by microsomal vitamin K epoxide reductase in dithiotreitol presence) CN Naphth[2,3-b]oxirene-2,7-dione, 1a,7a-dihydro-1a-methyl-7a-(3,7,11,15tetramethy1-2-hexadecen-1-v1)- (CA INDEX NAME)

L7 ANSWER 3 OF 9 ZCAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1989:610334 ZCAPLUS

DOCUMENT NUMBER: 111:210334

ORIGINAL REFERENCE NO.: 111:34779a,34782a

TITLE: Diagnostic importance of vitamin K1 and its epoxide

measured in serum of dogs exposed to an anticoaculant

rodenticide AUTHOR(S): Mount, Michael E.: Kass, Philip H.

CORPORATE SOURCE: Sch. Vet. Med., Univ. California, Davis, CA, 95616,

American Journal of Veterinary Research (1989), SOURCE:

50(10), 1704-9

CODEN: AJVRAH: ISSN: 0002-9645

Journal DOCUMENT TYPE: LANGUAGE: English

Administration of vitamin K1, s.c., to anticoagulant-poisoned (diphenadione) dogs provided diagnostic information within 4 h, when vitamin K1 and its epoxide were measured in canine sera. Twelve dogs (2 groups of 6) were given 2.5 mg of diphenadione/kg for 3 days. Dogs were treated with vitamin K1, 2.5 or 5 mg/kg/day s.c. for 21 days, and their responses were compared. Four nonexposed control dogs were given 5 mg of vitamin K1/kg/day. Serum concentration of vitamin K epoxide was significantly higher in diphenadione-exposed dogs than in control dogs 1 to 4 h after the initial vitamin K1 treatment on day 4. Vitamin K epoxide/vitamin K1 ratios were similarly higher and became more distinct. Cessation of vitamin K1 therapy on day 24 resulted in prolongation of one-stage prothrombin times in diphenadione-exposed dogs, becoming clearly evident on day 27. Serum vitamin K1 concns. were not detectable on day 27 in diphenadione-exposed dogs, whereas serum vitamin K1 concns. were readily detectable in control dogs. One-stage prothrombin time changes, during days 24 to 32, indicated 5 mg of vitamin K1/kg provided better protection than did 2.5 mg of vitamin K1/kg. Coagulopathy in the dogs was

25486-55-9

resolved by day 32. RL: BIOL (Biological study)

(of blood serum, in diphenadione-poisoned dogs treated with vitamin K1) 25486-55-9 ZCAPLUS RN

Naphth [2,3-b] oxirene-2,7-dione, 1a,7a-dihydro-1a-methyl-7a-(3,7,11,15-CN tetramethv1-2-hexadecen-1-v1)- (CA INDEX NAME)

L7 ANSWER 4 OF 9 ZCAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1984:453769 ZCAPLUS DOCUMENT NUMBER: 101:53769

ORIGINAL REFERENCE NO.: 101:8351a,8354a

TITLE: Formation of 3-hydroxy-2,3-dihydrovitamin K1 in vivo: relationship to vitamin K epoxide reductase and

warfarin resistance

AUTHOR(S): Preusch, Peter C.; Suttie, John W.

CORPORATE SOURCE: Coll. Agric. Life Sci., Univ. Wisconsin, Madison, WI,

53706, USA SOURCE: Journal of Nutrition (1984), 114(5), 902-10

CODEN: JONUAI; ISSN: 0022-3166

DOCUMENT TYPE: Journal LANGUAGE: English

English 3(2)-Hydroxy-2,3-dihydrovitamin K1 (I) [82162-05-8] was isolated and identified by comparison of its UV, mass spectra, and high-performance liquid chromatog. (HPLC) retention times with those of synthetic stds., and by its characteristic conversion to vitamin K1 quinone on treatment with the base triethylamine. I is formed from the vitamin K1 epoxide [25486-55-9], not from the vitamin K1 quinone, and can represent up to 3.5% of the dose and 13% of hexane-extractable metabolites present in liver 1 h after injection of 330 ug vitamin K1 epoxide/kg body weight It is formed in both normal and warfarin [81-81-2]-resistant rat strains, but to a significantly greater extent in the latter. Unlike the I formed by warfarin-resistant rat liver microsomes in vitro, the metabolite formed from racemic vitamin K epoxide in vivo was not optically active, nor was its formation inhibited by coumarin anticoagulants under conditions that completely blocked vitamin K epoxide reduction in vivo. On this basis, I formation in vivo differs from its formation in vitro; it is not a product of vitamin K epoxide reductase in vivo, but of some other possibly nonenzymic reaction.

RL: BIOL (Biological study)

(hydroxydihydrovitamin K1 formation from, by rats, warfarin resistance in relation to)

RN 25486-55-9 ZCAPLUS

25486-55-9

CN Naphth[2,3-b]oxirene-2,7-dione, 1a,7a-dihydro-1a-methyl-7a-(3,7,11,15-tetramethyl-2-hexadecen-1-yl)- (CA INDEX NAME)

L7 ANSWER 5 OF 9 ZCAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1981:528280 ZCAPLUS DOCUMENT NUMBER: 95:128280

ORIGINAL REFERENCE NO.: 95:21435a,21438a

ORIGINAL REFERENCE NO.: 93:21433a,21430a

TITLE: Chemical model studies for the mechanism of vitamin K

epoxide reductase
AUTHOR(S): Silverman, Richard B.

CORPORATE SOURCE: Dep. Chem., Northwestern Univ., Evanston, IL, 60201,

OURCE: Dep. Chem., Northwestern Univ., E USA

SOURCE: Journal of the American Chemical Society (1981),

103(19), 5939-41 CODEN: JACSAT; ISSN: 0002-7863

DOCUMENT TYPE: Journal

DOCUMENT TYPE: Journal LANGUAGE: English

Be Chemical studies for the mechanism of vitamin K epoxide reductase proposed previously are described using 2,3-dimethyl-1,4-naphthoquinone 2,3-epoxide (I) as a model for vitamin K epoxide. Treatment of I at room temperature with ethanethiol under acidic or basic conditions gives 2,3-dimethyl-2-ethylthio-3-hydroxy-2,3-dihydro-1,4-naphthoquinone (II), the product of epoxide ring opening by ethanethiol. The reaction of II at room temperature with Na ethylthiolate resulted in the rapid formation of 2,3-dimethyl-1,4-naphthoquinone, the reductive elimination product, and diethyldisulfide. These model reactions suggest that the enzyme-catalyzed mechanism proposed previously is quite plausible.

IT 25486-55-9

RL: PRP (Properties) (model for)

RN 25486-55-9 ZCAPLUS

CN Naphth[2,3-b]oxirene-2,7-dione, 1a,7a-dihydro-1a-methyl-7a-(3,7,11,15-tetramethyl-2-hexadecen-1-yl)- (CA INDEX NAME)

L7 ANSWER 6 OF 9 ZCAPLUS COPYRIGHT 2009 ACS on STN ACCESSION NUMBER: 1971:73514 ZCAPLUS

DOCUMENT NUMBER: 74:73514
ORIGINAL REFERENCE NO.: 74:11886h,11887a

TITLE: Vitamin K activity of phylloquinone oxide

AUTHOR(S): Bell, Robert Gale; Matschiner, John T.

CORPORATE SOURCE: Dep. Biochem., Univ. Rhode Island, Kingston, RI, USA

SOURCE: Archives of Biochemistry and Biophysics (1970), 141(2), 473-6

CODEN: ABBIA4; ISSN: 0003-9861

DOCUMENT TYPE: Journal LANGUAGE: English

GI For diagram(s), see printed CA Issue.

AB The biol. activity of phylloquinone oxide in vitamin K (phylloquinone) (I)-deficient rats was approx. the same as that of I. In warfarin-treated animals, I was an effective anticoagulant antagonist, whereas the oxide was not unless it was administered 15 min before warfarin. Warfarin markedly changed the metabolism of I, resulting in a large preponderance

of the oxide in the liver of anticoagulant-treated animals, suggesting that I oxide, because of structural similarity, may be an inhibitor of I and that warfarin exerts its anticoagulant effect by causing accumulation of the oxide.

IT 25486-55-9

RN

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BJOL (Biological study) (vitamin K activity of, warfarin treatment in relation to)

25486-55-9 ZCAPLUS

CN Naphth[2,3-b]oxirene-2,7-dione, 1a,7a-dihydro-1a-methyl-7a-(3,7,11,15-tetramethyl-2-hexadecen-1-yl)- (CA INDEX NAME)

L7 ANSWER 7 OF 9 ZCAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1960:63791 ZCAPLUS

DOCUMENT NUMBER: 54:63791

ORIGINAL REFERENCE NO.: 54:12357a-c

TITLE: Effect of progesterone on estrogen-induced hypercalcemia in the sexually immature pullet

AUTHOR(S): Wright, L. A.; Maw, W. A.; Common, R. H.

CORPORATE SOURCE: Univ. Montreal

SOURCE: Canadian Journal of Animal Science (1959), 39, 137-44

CODEN: CNJNAT; ISSN: 0008-3984

DOCUMENT TYPE: Journal

LANGUAGE: Unavailable

AB Sexually immature crossbreed pullets were given daily intramuscular injections of 0.5 mg. estradiol benzoate (I) for 14 days. Injection of 0.025-0.10 mg. of progesterone (II) did not affect the hypercalcemia induced by I but these levels of II augmentated the increase of oviduct weight induced by I. Injection of 0.5 mg. II significantly increased the hypercalcemia, but had no effect on oviduct weight induced by I. Hypercalcemia induced by 0.5 mg. I daily for 14 days was not affected by 0.05-0.10 mg. II daily, even though these levels of II significantly increased the augmentated oviduct weight induced by I. Injection of 0.5 mg. II daily increased the I-induced hypercalcemia significantly. As the dosage of II increased oviduct weight increased to a maximum and then declined.

(Derived from data in the 6th Collective Formula Index (1957-1961))

RN 25486-55-9 ZCAPLUS

CN Naphth[2,3-b]oxirene-2,7-dione, 1a,7a-dihydro-1a-methyl-7a-(3,7,11,15-tetramethyl-2-hexadecen-1-yl)- (CA INDEX NAME)

L7 ANSWER 8 OF 9 ZCAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1955:20717 ZCAPLUS

DOCUMENT NUMBER: 49:20717

ORIGINAL REFERENCE NO.: 49:4105a-b

TITLE: Vitamin K and new coagulation factors

AUTHOR(S): De Nicola, Pietro CORPORATE SOURCE: Univ. Pavia, Italy

SOURCE: 3a Giornata sci., Consiglio nazl. richerche, Convegno

vitamine, Milan (1953), (Suppl. to Ricerca sci., Anno

23), 754-9 Journal

DOCUMENT TYPE: Journal LANGUAGE: Italian

AB Kappa factor and factor VII prepns. obtained from blood after dicumarol treatment are less active than prepns. from untreated animals.

Vitamin K1 and its oxide can counteract this effect of dicumarol.

IT 25486-55-9, 1,4-Naphthoquinone,

2,3-epoxy-2,3-dihydro-2-methyl-3-phytyl-

(as coagulation factor)

RN 25486-55-9 ZCAPLUS

CN Naphth[2,3-b]oxirene-2,7-dione, la,7a-dihydro-la-methyl-7a-(3,7,11,15-tetramethyl-2-hexadecen-1-yl)- (CA INDEX NAME)

L7 ANSWER 9 OF 9 ZCAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1944:29408 ZCAPLUS DOCUMENT NUMBER: 38:29408

ORIGINAL REFERENCE NO.: 38:4313i,4314a

TITLE: Treatment of dicoumarol-induced

hypoprothrombinemic hemorrhage with vitamin K1 oxide

AUTHOR(S): Lucia, S. P.; Aggeler, P. M.

Proceedings of the Society for Experimental Biology

and Medicine (1944), 56, 36-7

CODEN: PSEBAA; ISSN: 0037-9727

DOCUMENT TYPE: Journal LANGUAGE: Unavailable

SOURCE:

AB In a human subject, hypoprothrombinemia and the secondary hemorrhagic phenomena induced by multiple large doses of dicommarol appeared to be corrected by the intravenous injection of a single dose of 500 mg. of vitamin K1 oxide. After a latent period of 4 hrs. there was a marked

elevation of the prothrombin level, but full recovery required about 5 days.

IT 25486-55-9, 1,4-Naphthoquinone, 2,3-epoxy-2,3-dihydro-2-methyl-3-phytyl-

(hypoprothrombinemic hemorrhage treatment with)

RN 25486-55-9 ZCAPLUS

CN Naphth[2,3-b]oxirene-2,7-dione, 1a,7a-dihydro-1a-methyl-7a-(3,7,11,15-tetramethyl-2-hexadecen-1-yl)- (CA INDEX NAME)

=> s 16 and lesion 54557 LESION

L8 0 L6 AND LESION

=> s 16 and lesions

107892 LESIONS L9 1 L6 AND LESIONS

=> d 19 ibib abs

L9 ANSWER 1 OF 1 ZCAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1943:42537 ZCAPLUS

DOCUMENT NUMBER: 37:42537

ORIGINAL REFERENCE NO.: 37:6717b

TITLE: Medical progress. Skin changes of nutritional origin

AUTHOR(S): Jeghers, Harold

SOURCE: New England Journal of Medicine (1943), 228,

678-86,714-23

CODEN: NEJMAG; ISSN: 0028-4793

DOCUMENT TYPE: Journal

LANGUAGE: Unavailable

B The diseases discussed include carotenemia, phrynoderma, keratosis follicularis, ichthyosis, Sjogren's syndrome, cheilosis, angular stomatitis, purpura of vitamin-deficiency origin, palmar erythema and

"dyssebacia." 167 references.

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http://www.nlm.nih.gov/pubs/techbull/nd08/nd08 medline data changes 2009.html.

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This file contains CAS Registry Numbers for easy and accurate substance identification.

See HELP RANGE before carrying out any RANGE search.

=> s vitamin k1 oxide 149527 VITAMIN 8986 K1 157467 OXIDE

L10 91 VITAMIN K1 OXIDE

(VITAMIN(W)K1(W)OXIDE)

=> s 110 and dermatol? 38526 DERMATOL?

L11 0 L10 AND DERMATOL?

=> s 110 and dermat? 142692 DERMAT? L12 0 L10 AND DERMAT?

=> s 16 and dermat?

87 L3 3653220 PY > 2003 142692 DERMAT?

L13 0 L6 AND DERMAT?

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